United States Public Health Service has established plants on the Texas border where baggage and effects may be disinfected and persons bathed to free them from body lice. Senior Surgeon C. C. Pierce, of the Public Health Service, who is now in charge of this work, says, among some rules recently published: "If a person infested with lice develops typhus fever he should be thoroughly disinfected, by having his hair clipped short and the body bathed with hot water and soap. All clothes should be boiled or destroyed by burning, care being taken that no lice that might be on the clothes escape." And "In order to rid a person's head of lice the hair should be soaked with a mixture of equal parts of kerosene oil and vinegar, covering the head with a towel for about one-half hour. The vinegar loosens the nits and the kerosene oil kills the adult lice. After one-half hour the head should be thoroughly washed with soap and water. Where the hair is very thick and where there are many lice more than one application of this remedy is necessary," and "in cases of children or men infested with head lice it is best to clip the hair and then wash the scalp with soap and water. This will be sufficient. The hair removed should be collected on a newspaper, rolled up and burned." Grubbs, in the Public Health Reports of October 20, 1916, recommends a rather more extensive method than this and uses a gasoline-soap spray and shower bath for the body and vacuum-cynanide process for the clothing and baggage, and in the British Medical Journal of June 19, 1915, may be found a short but comprehensive report entitled "An investigation of the best methods of destroying lice," by Kinloch.

At the present time we believe that the only agency for the transmission of typhus fever is the louse—particularly pediculi vestementi and possibly pediculi capitis. Body lice, or as sailors and soldiers call them, "seam-squirrels," live most of the time, and lay their eggs, on the clothes, but feed on the wearer. Their existence is comparatively short if deprived of food and warmth, and it takes their eggs about eight days to hatch out; therefore clothing frequently changed, and thoroughly laundered by boiling and ironing between changes, eliminates the danger of the body louse on one's person. A person sick with typhus cannot convey it if there are no lice about, and so, with this knowledge in our possession we can, if we care to be laconic, reduce the description of the quarantine measures against typhus fever to just four words—simply, no lice, no typhus.

This is an outline of how quarantinable and other diseases are prevented at ports of embarkation and on the borders—it is an outline of our defense against disease invasion—it shows briefly the state of our preparedness against medical assaults from foreign shores. The defense is not perfect—it might be improved upon, but such as it has it gives and no man can even begin to estimate the saving it occasions to our country, from a financial standpoint—the suffering it obviates from a physical standpoint and the mental sorrow and grief that are not, but might be, if these defenses do not hold out.

ACUTE POLIOMYELITIS WITH SPECIAL REFERENCE TO MYOCLONUS.*

By BERNARD OETTINGER, M. D., Long Beach, Cal.

The conception that infantile paralysis represents an inflammation of the anterior horns of the spinal cord gave place to one that other gray elements of this organ were also implicated and this view to the recognition of an infection involving the entire cerebrospinal axis. Hence some authorities now prefer the designation polioencephalomyelitis. Although this title is descriptively more correct, the older term poliomyelitis is herein employed because less unwieldy.

Beyond a better understanding of its pathology our clinical conception of infantile paralysis too, has grown. But one form, the spinal type, was recognized until most recent years. Only since 1911 have types been identified compatible with the idea of a general infection of brain and cord, (together with enveloping membranes), yet with varying dominant features relating to one or another unit of the cerebrospinal axis. This modern view has been the direct result of careful studies in recent epidemics but the knowledge so gained may be applied to sporadic cases. Here, too, infantile paralysis encountered as a seeming meningitis or encephalitis or in the guise of a profound general infection without paralysis, has usually been otherwise clinically designated. Recognition of the last group, the so-called abortive type, is of great import from the viewpoint of incidence alone, being estimated at from 15% to 60% of all cases in some late epidemics. Standard text books consider our added knowledge in so far, but not even these take cognizance of early motor phenomena in poliomyelitis which may present. The latter deserve comprehensive clinical study as an aid in the diagnosis of abortive cases and connotatively, perhaps, of the preparalytic stage of classic spinal types. Two case reports are herewith submitted in respect to diagnostic problems thus suggested.**

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* Read before the Long Beach Branch of the Los Angeles County Medical Society, November 24, 1916.

** Although Flexner and Noguchi identified the causal organism of infantile paralysis in 1913, a crux in diagnostic difficulties as regards sole reliance upon bacteriological findings, has lain in the inability to recognize the bacterium with ordinary laboratory equipment because of its infinitesimal size. Almost a decade previously Scandinavian investigators had isolated a diplococcus from tissues of fatal cases and produced characteristic paralysis with cultures thereof in injected animals. These findings however were, on the whole, not confirmed. The entire question has now been reopened by American observers. September 30th of this year, Mathers [1] reported finding a gram positive microorganism in brain and cord tissue and mesenteric lymph nodes obtained from fatal cases. Corroboration followed in work done under the Mayo Foundation [2] and from the Pathological Laboratories of the Cook County Hospital.[3] Nuzum and Herzog found the microorganism, a streptococcus, in the spinal fluid of eight out of nine cases and in a later report [5] in forty-five out of fifty cases. That this microorganism is either the carrier of the real ultra-microscopic virus or what is more probable that

Case 1. A girl aged four years is seen in consultation on the fourth day of illness in bed. For a week, while up and about, gastrointestinal symptoms have been present. In dorsal decubitis the patient lies with both legs slightly flexed. She cries when these are handled, resists their complete extension but later when asked to kick down the covers, she fully extends the lower limbs. She does not sit nor stand on request but when held upright, again holds both legs flexed with toes just touching the bed. The arms are used normally. The child whimpers a good deal. The psyche is not involved, her attention being frequently engaged to tell what she would like to eat, how she would like to take a ride, etc. There has been no vomiting, headache nor convulsions. A range of fever from 99½ to 101 has persisted since in bed. At this time the skin is clear. (A few days later a macular rash appeared on the chest.) Both pupils react to light; there is slight photophobia but the conjunctivae are not reddened. No cranial nerve paralysis. Marked stiffness of the neck presents and movement of the head is painful but neither at this time nor later was the latter drawn back. An occasional ralê can be heard in the left lower lobe. At my first visit the presence of the right abdominal reflex is doubtful and the right patellar reflex can just be elicited. Thereafter I can elicit neither abdominal nor patellar reflexes. There is some but not marked hyperaesthesia of the skin. With the patient's attention otherwise attracted, the soles of feet can be stroked without pain. The legs are not, and later did not become paralyzed.† On holding either leg in my hand, I experience slight rigidity, a condition entirely at variance with the traditional thought of flaccid or hypotonic musculature in infantile paralysis. With this occurs a peculiar fibrillary tremor under the fingers. Upon my second visit two days later the child seems not so bright. Otherwise the symptoms are as before with this further exception. A brief examination now excites the patient. She cries violently and directly shocklike jerks of the head and extremities recur at short intervals. The child continues to cry and its distress is impressive.

The following conditions were considered, viz.: acute rheumatism, osteomyelitis, meningitis (tuberculous or meningococcic) and poliomyelitis. Absence of redness and swelling and the fact that the joints were not involved ruled out acute rheumatism which condition would also have developed greater temperature. Palpation showed the epiphyses of the long bones not particularly tender which excluded osteomyelitis or epiphysitis due to other causes than septic infection. The fact that pain was not severe when the lower limbs were at rest spoke against all these conditions. A point noted was that all symptoms with the single exception of the neck stiffness referred to meningeal involvement of the lumbar region. Tuberculous meningitis might be excluded in the face of acute development of nervous symptoms yet absence of headache and vomiting. Also, because acute tuberculous meningitis is a basal affair some involvement of the cranial nerves would have been a likely occurrence. The same factors spoke against a meningococcic infection. Examination of the spinal fluid at the County Hospital definitely ruled out this entity. This procedure was denied us while the patient was at home, but the continued mental clarity, absence of cranial nerve involvement, localization of symptoms pointing to inflammation of the spinal meninges alone, together with the vanishing abdominal and patellar reflexes determined a diagnosis of acute poliomyelitis.

Case 2. A boy of 2½ years. A maternal grandmother died of osteomalacia. Father and mother well but the latter has an enlarged thyroid and slightly bulging eyes. Of the mother's family one brother died at 43 years of tuberculosis, one brother at 30 years of exophthalmic goitre, two sisters in infancy and three sisters and two brothers are living and said to be well. The mother is married seven years; no miscarriages. Two older children are well; these and the patient born at full term. All labors long and hard but accomplished without the use of instruments. During each pregnancy much vomiting up to and during parturition. Patient was well nourished at birth. The mother, who is intelligent, says that at three months the child had a fever; was fretful and restless. Soon after attacks passed through the body which would stiffen. When the spasm relaxed, the glottis opened and there was a crowing sigh. Following the spasm the left arm was held rigid for a time. After a few days a series of yet harder attacks of like character occurred. There was much sweating about the head. Now supervened short attacks in which the patient would lie very quietly, staring vacantly and then occurred quick jerking of the head and extremities. Following these paroxysms he could not be quieted for some time. At present he eats much and anything offered him. Bowels and bladder act normally.

Physical examination: The patient is in dorsal decubitus. The head in general is well shaped but the occiput is flat. The face is exceptionally handsome, the expression intelligent. It is said the child can speak a few words. The body is long; the legs and arms thin; the fingers noticeably slender but withal there is no emaciation. The

the easily visible coccus is the aerobic form and the ultramicroscopic bodies the anaerobic form of the same organism reflects, in general, conclusions held. Dixon,[4] however, states that studies undertaken by him in 1907 and 1910 resulted in finding this gram positive diplococcus in secretions from nose and throat and in culture from the spinal fluid in acute poliomyelitis cases. But the results of cultural injections of animals lead him to believe that although the constant presence of the diplococcus shows it has something to do with the causation of the disease and may be symbiotic in its relation to the principal agent, this germ is nevertheless not the chief etiological factor.

† However, after three weeks with the patient in dorsal decubitus there is, without paralysis, slight relaxation of the left peronei muscles.

skin is of good color, fine and without a blemish. The left ear is smaller and the left eye slightly lower than are the same on the right side. The left bony thorax is somewhat smaller than upon the opposite side. Exact measurements of arms and legs are not made but there is no marked difference in their length. Patellar reflexes present; Babinski positive (probably of no moment in this instance). Abdominal reflexes absent. No paralysis of cranial nerves or those of the extremities but the musculature in general is hypotonic. When placed in a sitting position and held, as the patient cannot sit up without support, the head is held erect but the spine in the lower dorsal region bows with a posterior convexity and the abdominal muscles bulge laterally. There is no titubation of the head. Resonance is found over the upper sternal region (absence of thymus?). Chest and abdomen negative. The testicles are undescended.

At a subsequent visit the following events were noted while the patient was sleeping in his basket. The eyes are opened and the bulbi moved slightly. Then the head, arms and legs jerk in quick sequence. The child awakes, cries, is restless and directly athetoid movements of one hand and then the other are noted. Some time elapses before the babe is quieted. These attacks are said to be frequent and always uniform.

A full discussion of this case would carry us too far afield from the subject in hand. Suffice it to say in passing that if we view the laryngismus stridulous, spasmophilia in general, the extreme body length and sternal resonance in the thymus region as evidence of probable thymic involvement, we note occurrence of abnormal ductless glands in three generations of this family. Even so, however, this may mean no more in the present instance than lessened resistance to an infection which certainly initiated the patient's illness. The short time the patient was under observation he was given small doses of thymus gland without appreciable effect. No doubt also with the idea of improving calcium metabolism the family physician (in another state) had given the patient up to 36 grains of calcium chloride a day for weeks with no result beyond decided stomachic disturbance. The history of fever and epileptoid attacks at once suggested an encephalitis but the residual paralysis of abdominal muscles and spinal erectors in the lumbar region discover the true nature of the infection, viz.: acute infantile paralysis.

In considering the shocklike motor phenomena of these cases one recalls that poliomyelitis aside from paralysis presents the picture of meningitis or its counterpart due to intense hyperemia of the brain occurring in some severe infections. Again, motor symptoms have not been unremarked in meningitis or cerebral hyperemia as witness the "startings" of tuberculous meningitis referred to by Osler and the generalized tonic contractions of the muscles of the jaw, neck, back and limbs noted by Escherich usually as a sequel of some acute infection or occurring as an independent malady. However, such symptoms have been interpreted (and no doubt correctly), as toxemic irritation of the motor cortex and have not been regarded as of specific diagnostic significance. For this reason Colliver's[6] comments upon the diagnostic import of early motor phenomena in infantile paralysis is of considerable interest. He has remarked these in sixteen cases during the preparalytic stage. His observations are convincing in respect to variety of motor phenomena noted but they fail to give, as I believe, entirely concrete impressions of movements seen.‡ I put to one side the tremor noted in case 1, being doubtful if it should be classed in the category which Colliver depicts and indeed, in the same respect, I am not entirely certain regarding the dramatic and shocklike jerking of head and limbs which was common to both cases. Yet it would seem this may be identified as "twitching which may affect the whole body and in the beginning lasts less than a second." In any event, muscular spasms of this character occurred in both instances during the acute stage of the disease and once as a residual symptom. The phenomenon comprehends a typical *myoclonus*. This symptom has been defined as "involuntary, unsystematized, arrhythmic, quick, muscular contractions similar to that produced by an electric shock. They may be localized or disseminated and may embrace a muscle, a muscle group or only a few fibres" (Church and Peterson). In this connection we recall that myoclonus sometimes occurs with epilepsy comprising the "association disease" myoclonus—epilepsy, and again, that between myoclonic paroxysms, tremor of the same muscles may present sometimes fibrillary in character (so-called live flesh). It is also true that myoclonus is best known as

‡ Colliver says: "The symptom referred to is a peculiar twitching, tremulous or convulsive movement of certain groups of muscles lasting from a few seconds to less than a minute. The amplitude of vibration is greater than a tremor, not so constant and long as a convulsion and more regular than mere twitching, yet it has some elements of all of these. It usually affects a part or whole of one or more limbs, the face or jaw, but it may sometimes affect the whole body. The symptom may readily be overlooked in the beginning as it usually lasts less than a second and unless the patient is disturbed does not recur oftener than every hour or so. Later, the duration of the spells lengthens to a few seconds, recurring also at shorter intervals. This condition is often accompanied by a peculiar cry similar to the hydrocephalic. At times there is a slight convulsive movement "just like a chill," as the mothers say, during which time the child is apparently unconscious with eyes set for a few seconds and then he apparently becomes perfectly normal again. This brief unconsciousness during which the child's eyes are set, may occur without noticeable convulsive movements. It acts thus something like a petit mal. I have observed it as a twitching of the lips with tongue running in and out and a working of the jaw, preceding bulbar cases. . . . The least stimulation of the skin is followed by slight convulsive movements with rigidity of the arm, fingers separated and wrist flexed (athetoid movements?). When the patient turns in bed, through either an external stimulus or an effort to coordinate, the movements are quick and jerky accompanied usually with slight convulsive movements of the limbs. The least noise produces in certain cases short series of convulsive movements similar to those in strychnine poisoning only not so general. This symptom seems to be similar to the infection neuroses described by neurologists of which tetany and chorea are good examples." This description covers a wide latitude of motor phenomena, viz.: a range of muscular contraction from tremor to convulsive movement of the whole body, and spasm both tonic and clonic. Colliver states that practically no reference to this symptom can be found in the literature of poliomyelitis but that both Zappert and Wilbur noted muscular twitching in the limbs. **Jerking** of the limbs and head better describes the particular movements observed in the cases here recorded.

a clinical entity, acute or chronic, of unknown etiology and that it has the tendency to subside after many months during which remissions and exacerbations may have been experienced. However, what is of particular interest to the subject in hand, is the precedent observation of myoclonus in acute infectious disease, viz.: in Dubini's disease, otherwise known as *electrical chorea.* The meager epidemiology of this affection records a 90% fatality. First described in 1845 in reference to cases which occurred in malarial districts of Italy, it was for a time thought to be of paludal origin. Its etiology, however, has remained obscure. So much as is known of the pathology of electrical chorea comprises "pulmonary and splenic congestion, inflammation of the meninges, increase of cerebro-spinal fluid, cerebral congestion especially at the base and softened foci in the cortex and great ganglia (Church and Peterson).[7] The brief clinical descriptions available refer to rythmical movements, as if from an electric shock, in the extremities and rarely in the head and face. Fever may be present. Pain in the head and neck may be an early symptom. Sensibility is not greatly affected but hypersensitiveness may easily be evoked and this exalts the motor phenomena. Epileptiform attacks may occur. In some cases paralysis may supervene and toward the end of the attack atrophy of muscles may be apparent (Church and Peterson; McCarthy[8]). The foregoing suggests the interesting possibility that infectious electrical chorea is really acute poliomyelitis with myoclonus as a dominant feature. The further study of motor phenomena which may appear early in acute poliomyelitis seems indicated. While doubtless in no way pathognomonic, such manifestations may prove a clinical aid at a time when diagnostic difficulty is the rule.

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WAR DEPARTMENT

HEADQUARTERS WESTERN DEPARTMENT

Office of the Department Surgeon

San Francisco, Cal.

April 19, 1917.

Dr. Sol Hyman,
Editor, California State Journal of Medicine,
Butler Building,
San Francisco, Cal.

Dear Doctor:

In anticipation of the early legislation by Congress to call five hundred thousand men at once and five hundred thousand more within the year into active training and service, and, in view of the necessity for the immediate enrollment of a large number of the younger medical men of the country in the Medical Reserve Corps for service with these troops as regimental surgeons and assistants, ambulance companies, field hospitals, etc., I would appreciate any publicity you may be able to give in your columns relating to this matter. Information and all necessary blanks can be obtained from me either at the Department Surgeon's Office or at the Letterman General Hospital.

Very truly yours,

Signed: GUY L. EDIE,
Colonel, Medical Corps,
Department Surgeon.

SYNTHETIC SUBSTITUTES FOR COCAIN WITHDRAWN FROM FEDERAL REGISTRATION.

On page 129, Department of Pharmacy and Chemistry, attention was called to the decision of the United States Circuit Court of Appeals, holding that synthetic substitutes for cocain and eucain did not come under the jurisdiction of the Harrison Act.

Below we print the letter of instructions of the Treasury Department in conformity with this decision:

B.C.K. TREASURY DEPARTMENT.

Office of Commissioner of Internal Revenue,
Washington.

March 28, 1917.

M-n. Mim. No. 1497. Suspending enforcement T. D. 2194, relating to synthetic substitutes for cocain.

To the Collectors Internal Revenue, Revenue Agents, and Others Concerned:

Referring to T. D. 2194, holding that any synthetic substitute for cocain, alpha or beta eucain, or their salts or derivatives, comes within the provisions of the Act of December 17, 1914, and that persons using or having in their possession any such synthetic substitute are required to register and obtain such substitutes upon official order forms and otherwise conform to this act, this office has decided to suspend the enforcement of the ruling of April 26, 1915, until you are otherwise advised.

This action is taken in view of the decisions of the U. S. District Court, Southern District of New York, of June 28, 1915, and of the Circuit Court of Appeals for the Second Circuit, of February 21, 1916, holding that these synthetic substitutes did not come within the provisions of section 1 of the act.

Therefore, you are directed to notify all registered persons in your district or others who may be affected by T. D. 2194 of the suspension of this ruling.

W. H. OSBORN,
Commissioner.

Approved:
W. G. MCADOO,
Secretary.